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BIOGRAPHY

SIR JOHN McFADYEAN

HISTORIANS of the future will say that the years between April 20, 1876, and February 1, 1941, were probably the most momentous in the story of the veterinary profession. It will be noted that the dates mark the times of the graduation and death of Sir John McFadyean. The period under review marks the progress of the profession from a feeble adolescence to a full and vigorous maturity. It was peopled by many sage and courageous leaders whose strenuous efforts to rectify the retrogressive effects of the Coleman era met with well-merited success. These men did not correct the mistakes of the past only, but they wisely found time to plan for the future. They envisaged how the profession could assume its rightful place in the State and in the public esteem by the virtue of a sound scientific educational system and by the attainments of its members. The present high status of the profession and the recognition which rightly it commands provide ample evidence of the success of well-laid plans. It was a fortunate circumstance that, during this critical period, there were men of ability at hand and men who were prepared to give of their time and sustenance for the cause. Now most of these men were just ordinary fellows such as can be found doing the work in any democratic body or institution. Amongst their number, as is always the case, there were men gifted with talent, vision and personality. They were the giants. There was one who from the very day that he graduated began to take such a deep interest in his profession, an interest which grew with the years, that in

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time he bestrode his contemporaries like a colossus. This man was John McFadyean. No man has commanded more respect from his fellows or influenced their destiny, scientific or political, to a greater extent. No one has brought greater renown to veterinary science. To many he was the great teacher, the austere schoolmaster, to all he was the undisputed leader. Most of us never spoke to him in a personal manner; it may be that our only contact outside the lecture room was to greet him as he passed, yet by instinct we were aware that he knew all about us. Of his private life we know but little. We do know that he was blessed by a gracious and kindly partner and a happy family. We can be sure that his high ideals and character were not confined to his public life.

He made his own way in the world, for he had great ability and energy.

He was born on June 17, 1853. His father was one Andrew McFadyean, the tenant farmer of Barrachan of Bladnoch in Wigtownshire. He received his primary education at a local school, probably at the feet of one of those renowned Scottish characters, "The Dominie." From this he passed to the Ewart Institute at Newton Stewart, a secondary school at which he was able to prepare for admission to the University or to a profession.

He left school at the age of 16, for it was intended that he should help and later follow his father on the farm. The farm was a mixed one, and he gained a practical knowledge of all types of agriculture which was to stand him in such good stead in later years. Life on a Scottish farm is never easy, and it is said that he worked early and late and as hard as any of the paid servants. He never lost this capacity for hard, unremitting toil. Farming prospects in the 1860's were not good, and young McFadyean decided to abandon the life and become a veterinary surgeon. In making this decision he was influenced by the local practitioner, a Mr. John Doig. In October, 1874, he entered the "Dick" Veterinary College as a pupil. Professor Walley, the pioneer of veterinary public health service, was the Principal. The course of instruction covered a minimum of two years. At the end of that period he obtained the Certificate of the Highland Agricultural Society and the Diploma of the R.C.V.S. At that time it was the custom for Scottish students to take the Highland Certificate. It granted a licence to practise in Scotland.

His student career must have been creditable for records show that he gained the second prize in the Fitzwygram competition, and the gold medal of the Highland Society for the best knowledge of all subjects.

In the autumn of 1876 he joined the staff of the Dick College. He became the lecturer on Anatomy. His prospects appeared to be poor, for the Dick College was then in its worst period, students were few and equipment miserable or absent. The sole income of the College was derived from students' fees and from the clinical practice. However, the appointment had its compensations, for it brought him in contact with learned men, some of whom were

also attached to the University. He exhibited a great keenness in the search for knowledge, and while continuing his duties at the College he attended classes in Science and Medicine at the University. He was noticed and encouraged by such men as Professors Turner, Chiene, Greenfield, Dixon and Geikie. These great men gave him every assistance. In due course, in 1882, he obtained the degrees M.B., C.M. and B.Sc. In this way he obtained the scientific training and turn of mind and built a sure foundation for his future career. It also served to make him more conscious of the shortcomings of the system of veterinary education at that time. There is no doubt that this awakening had a most profound effect on his whole outlook, and was the most potent factor which guided and strengthened him in his efforts to improve the curriculum in the Veterinary Schools.

He continued in his post at the Dick College, and in 1883 he married. His wife was the daughter of Principal Walley. His finances did not improve to a degree commensurate with the new responsibilities, and a search for additional income was necessary. He put up his plate as a medical man. It was not a success, for at heart he was a veterinarian. There can be no doubt that it might have been a success if he had been prepared to devote his ability and energies to the quest of financial gain rather than to the duties of his academic post.

He decided to devote all his attention to Veterinary Science, the plate was withdrawn, and he continued as Lecturer in Anatomy at the Dick College until 1892.

During this period he compiled and published the well-known Dissection Guide to the Anatomy of the Horse, and the smaller work on Osteology and Arthrology. The latter was named Part I of the Comparative Anatomy of the Domesticated Animals. Both books soon became standard textbooks, and have continued to be held in such regard right up to the present day. It is to be regretted that he did not complete the three volumes which were originally intended. At this time the science of Pathology was steadily maturing, and McFadyean was attracted. He taught Histology and so, possessing a sound knowledge of normal structure, it was perhaps natural that he should be interested in the variations which were incidental to disease.

In 1883 he instituted a course of instruction in Pathology at the Dick College. It was an additional and voluntary task, for which he received no remuneration. In 1886 the Edinburgh Pathological Club was founded. He was one of its first members and through it he came in contact with the great pioneers, amongst whom were Sims Woodhead, Alexander Bruce, Noel Paton and Robert Muir. We have it from Sir Robert Muir that the atmosphere of this Club was one of keen inquiry. There can be no doubt that McFadyean took full advantage of all that passed, so that by the year 1892, when he left Edinburgh, he was already recognised as a competent Pathologist.

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He formed a great friendship with Sims Woodhead, and in collaboration they presented a paper to the fourth Annual General Meeting of the N.V.M.A. The meeting was held in Edinburgh, and the title of the paper was " Notes on some Microparasites of the Domesticated Animals."

Realising that English veterinary literature was sadly deficient from the Pathological point of view, he set himself to acquire a knowledge of the French and German languages in order that he might be familiar with the work of continental investigators.

In 1888 he founded *The Journal of Comparative Pathology and Therapeutics.* It was a private enterprise, and he remained in sole control until 1940, i.e., for over 50 years, a unique achievement.

McFadyean's interest in Pathology was not confined to the purely academic field. He sought to put the science to everyday use. At a very early date he applied its teaching to the realms of meat and milk hygiene. This ability to apply the lessons of the laboratory to a practical use was typical of the man. There is no doubt that his practical training on the farm and his association at an early age with distinguished men of science gave him an extraordinarily broad outlook. It can be said that he made Pathology a useful subject.

These years of hard work and the fortunate possession of a first-class brain stamped him as a man who was destined to attain high rank in his profession. When the call to London came, the Governors of the Royal Veterinary College could not have made a wiser choice. He was appointed to be Dean of the College and Professor of Pathology and Bacteriology. Sir George Brown continued as Principal for a short time, and on his retirement McFadyean was appointed Principal and Dean. He held the post until he retired in 1927, a period of 35 years. He came to an impoverished institution and, it is said, one in which discipline was at a low ebb. Those who have first-hand knowledge have told of the transformation which took place in the teaching and discipline. He did not succeed in placing either the buildings or finances in a sound state; indeed, their state deteriorated steadily, and their rehabilitation was a task which awaited his successor. Certainly he restored the scientific reputation of the institution to its proper place in the public esteem.

He developed the Pathological Department, extending its activities to suit the ever-growing and changing needs. Such funds as were available were wont to find their way to this department, whilst other sections of the College were left to suffer the pains of severe financial cramp. This state of affairs formed a perennial topic of conversation amongst those members of the staff who were not classified as pathologists. It remained only a topic of conversation, as did questions relative to salaries and conditions of service. By establishing post-graduate courses he showed the spirit of a true pioneer. As a lecturer he was superb. He possessed the great gift for imparting knowledge,

and he could make the subject live. He took full advantage of the wealth of material which was at hand, and in the midst of all his official activities found time for research.

For many years he was the only Veterinary Research Pathologist in this country, and his laboratory was the nursery of men who have themselves achieved fame. His work ranged wide, so wide that it amazes the modern investigator. Morbid Anatomy, Bacteriology, Protozoan and Virus diseases all came within his purview. He most certainly did not live in any watertight compartment; he was versatile, and probably it was this capacity to tackle almost any problem that came his way which accounted for his success. There is no doubt that the extreme degree of specialisation which is so prevalent in present days has many disadvantages. Its devotees never get a clear picture of the whole, they are apt to acquire a one-sided view, and in some cases become entirely divorced from their primary calling and its needs. His laboratory was a converted slaughter-house and poorly equipped according to modern standards, or even to the standards of his own time. He was a living proof of the saying: "It is the man and not the buildings which counts." As time went on he attracted pupils who, in turn, became disciples and went forth with the McFadyean hallmark, which was accepted as being of the highest standard in all parts of the world.

Not the least of his talents were those of a commentator or critic. His strong protests to the British Congress on Tuberculosis in 1901 when he alone had the courage to oppose the pronounced view of the great Koch would be sufficient to give him lasting fame. His opposition to Klein's views when anthrax was being debated, and his masterly closure of the "Lignieres" discussion in foot and mouth disease at the Eleventh International Congress in London in 1930 are other examples of his prowess as a critic. There must be few diseases of the domestic animals to which he has not given some attention and on which he has not contributed useful information.

It would, therefore, be impossible to review, and to do justice to, the scientific work and attainments of McFadyean in an article such as this. A review has already been made by a gifted and well-informed author in a contemporary publication. It is to be hoped that, in justice to McFadyean, this is only what might be called a preliminary note, and that the author in question will be persuaded to prepare a more extensive record at some future date.

Throughout his career McFadyean took a deep interest in the government and general welfare of his profession. He first came into note as the champion of the students in their efforts to promote reforms in the examination system. He felt that there were certain injustices, and reforms were urgently needed. The controversy waxed high, hard words were spoken; those in the citadel were not sparing in their criticisms of McFadyean. Undaunted, he carried on, for he had a good cause. The reforms were effected. He was

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elected to the Council of the R.C.V.S. in 1893, and was President in 1906-1909, again for a period in 1910, and finally in 1930, the year of the International Congress. An able debater, "a bonny fighter," a gifted administrator and of great integrity, it was not long before he was exerting a dominant influence in Council. It has been said that for many years "he was the Council." He fought to preserve the heritage of the profession, its independence, its right to self government and to elect its rulers from the ranks of its members. There are some who do not realise their debt to McFadyean. But for his unbending fight to preserve these democratic principles against all who would assail them, whether from within or without the profession, it is probable that the profession would be to-day simply the Cinderella of some more powerful body. There is no doubt that his attitude in these matters made enemies for him amongst interested persons outside the profession. He suffered attack, but he could always hit back, and to our great pride he was never beaten. His services on the Examination and Parliamentary Committees were particularly valuable at a time when the curriculum was under almost constant review and there was being enacted legislation which might affect the profession vitally. He retired from the Council in 1939, having served for the record period of 45 years.

For many years he was an active member of the N.V.M.A., contributing frequently to the discussions of the parent body, and also to its branches. He was honoured by his profession, by the Royal Society of Edinburgh, by the University of Aberdeen, by many Societies, by foreign countries and by his King.

He was elected Hon. Associate of the R.C.V.S., was the recipient of the Steel Memorial Medal, and was the first member to receive the Diploma of F.R.C.V.S. (H.C.).

He received the honour of knighthood in 1906, to the great delight of the profession, which marked its approval in no uncertain fashion. By 1906 Sir John was acknowledged by many as the leader of the profession, and the years which were to follow only served to establish him more firmly in that position. He became the man to whom all could turn for sound advice on the many problems associated with Veterinary Science. Nor was that trust misplaced for, in 1928, at the time of Sir John's retirement from the post as Consulting Veterinary Surgeon to the R.A.S.E., the late Earl of Northbrook said:

> His sound advice had always been at the disposal of the Council of the Society. His opinion had always commanded the greatest respect, and there were few, if any, men who had done more in recent years for the advancement of veterinary knowledge."

In his later years, as is often the case of great men, Sir John assumed somewhat of the powers of a dictator. However, his dictatorship was a benevolent one, and its sole aim was for the advancement of his beloved profession.

If such power had been his in early life the present story might not be told in the same vein. Fortunately, it came to him, and a glance at period photographs will substantiate this point, when the mellowing effects of a long and useful life had gained their way.

It is debatable if he did not delay his retirement to too late a date. One would wish that he had retired at the height of his glory and that he could have spent more years free from his many professional activities.

It is also to be regretted that he did not pass on his great wealth of knowledge of Veterinary Pathology in a form that could honour the shelves of the Veterinarians' library.

What manner of man was Sir John? A biographical note should make some attempt to give a picture of its subject. He was a relatively tall and spare man with broad but slightly rounded shoulders. Large of bone, his hands were strong yet fine and full of expression. His head was not large, his cheek bones rather high and somewhat pronounced. He had a noble forehead and a great dome. His eyes were well placed and appeared to have some difficulty in suppressing a look of quiet humour. As the years advanced this became more pronounced. Yet these eyes could look through and through a man. His voice was soft and higher than the average in tone. He had a command of good English, his speech being clear and distinct and its modulation that of a Southern Scot. He was partly bald, a feature which served to accentuate the fine design of his dome. The approach of grey hairs only contributed to the dignity of a fine head. When he came to London he possessed a fine flowing moustache, which underwent a progressive trimming or diminution to conform with changing custom. In his old age it was almost military. He walked with an easy stride, his head carried with a slight backward tilt. His clothes were always good, but it was only in his later years that they could be described as of fashionable design. He was probably the last Veterinary Professor to discard the traditional top hat. He never appeared to be in a hurry, yet so well did he order his goings that he was rarely late. To be interviewed by him was something of an ordeal for, although he was the kindest and most gentle of men and always took great care to put one at ease, one could not suppress the feeling of being in the presence of the Head.

His official life was ruled by the time-table and his laboratory efficiently organised. Yet if one stepped across the quadrangle to his private study a shock was imminent. On the first occasion that the writer had this experience he was bewildered. So great was the apparent chaos that one had difficulty in entering through the door. Papers and books were all over the place, piled in irresponsible heaps on chairs, tables, desk and floor. Sir John was writing, using the arm of his chair as a rest, for there was no room on the desk. However, the chaos was more apparent than real. It became necessary to refer to

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the contents of a letter. Sir John arose, went to a table, inserted his hand into the depths of a great pyramid of "higgledy-piggledy" papers, and in a moment withdrew the desired missive. It is said that by getting down on his hands and knees he could repeat the performance with the heaps on the floor. He seemed to lose all count of hours when in his laboratory after the official College hours. It is said that Lady McFadyean had to fetch him to meals or to bed on many occasions. Such was the Sir John whom we were privileged to know, the man who brought fame to an almost unknown profession, the man of whom Sir Francis Duck, speaking at a banquet of honour in 1906, said:

> "The work which Sir John has done in the realms of scientific research will live";

and in reply, here are Sir John's words:

> "I should be happy and absolutely content if the profession think that I have done my share either as a teacher or investigator in helping the profession."

Sir John died on Saturday, February 1, 1941. He was laid by only for a short time; he enjoyed full mental faculty until the end.

He must have passed on happy and contented, for the postulates which he enunciated in 1906 as being necessary to ensure that state had been absolutely fulfilled.

J. McC.

Obituary

WILLIAM BULLOCH, M.D., LL.D., F.R.S.

Prof. William Bulloch died on February 11, 1941. By his death the veterinary profession has lost the best friend it ever had in the sister profession of medicine. He was born at Aberdeen on August 19, 1868. His father, John Bulloch, was a copper-plate artist of repute, his brother was J. M. Bulloch, the author and renowned editor. He was educated at the local Grammar School and in due course proceeded to the University to study medicine. He graduated M.B., C.M., with highest honours in 1890. In 1894 he took his M.D. with similar honours and gained the Struthers gold medal and prize in anatomy. After he qualified he entered general practice as an assistant, but like his great friend Sir John McFadyean, he was not satisfied with the state of his knowledge. To his exact and inquiring mind and high ideals general practice was simply a life of "Mumbo Jumbo and platitudes." To quote his

WILLIAM BULLOCH AND HIS FRIEND "TIM."

own words when describing this period: "He felt that he had done nothing worth while save show some kindness."

Thus at an early date was the great spirit of Bulloch born, a spirit and will which was indefatigable in the search for knowledge and which could not tolerate pretence or humbug in any form.

In his own words: "He decided that there must be something to learn in the world." He had no influence or knowledge of the world beyond the confines of his native city, but being a true Aberdonian, he had saved £100. With this and ignorant of the language, he took passage on a cargo boat bound for the Continent. He landed at Hamburg and was able to attach himself to Birch-Hirchfeld as a voluntary assistant. He next proceeded to Vienna and studied under Politzer Caposi and others. How he made the financial ends meet and overcome all other disadvantages can only be attributed to his zeal and indomitable courage. What a lesson to modern youth, who looks contemptuously on scholarships of £250 per year. I believe that these days were amongst the happiest of his life. He certainly never regretted them and on occasion would relate these adventures with great gusto.

In due course he returned to Scotland by cargo boat, and on that very night made contact with his friend and fellow student, Arthur Keith. Keith persuaded him to go to London. At that time medical circles in London were very conservative, and things were difficult for those who did not possess the diploma of the Royal Colleges. Bulloch was told that there was no prospect without this diploma. He was lucky in being appointed assistant to David Ferrier at King's College. Later he became assistant to Victor Horsley, Professor of Pathology at University College. Horsley encouraged him and enabled him to study under Roux in Paris and Salomonsen in Copenhagen. His life was shaped towards bacteriology, and he knew personally all the great figures of the golden age of that subject. After serving for two years as bacteriologist in charge of the Lister Institute antitoxin laboratory at Sudbury, he was elected bacteriologist to the London Hospital. This was in 1897, and he held the post until retirement in 1934. In 1919 he became Goldsmiths Professor in Bacteriology in the University of London. On his official retirement he was appointed Consulting Bacteriologist to the London Hospital and Emeritus Professor of the University. Retirement was purely a release from official duty. He retained his room and laboratory at the Hospital and worked there daily, keeping the same regular hours as hitherto. He continued his work on the sterilisation of catgut with notable success. He made it safe. He made many valuable contributions to scientific literature, and he was a great historian. Appreciations of these have already appeared in the medical Press and it is the object of the writer to try to picture the man as we knew him.

Bulloch was a short, stout man who, at any rate in early and middle life, was of a somewhat pugnacious demeanour. He was a hard, methodical and

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exact worker. His life was regulated by the interests of his science. He was in love with his work. It also served as his recreation. Yet he was never the absent-minded professor. He was a great wit, full of full-blooded humour and the joy of life. His laboratory was a happy place to which a constant stream of visitors was attracted. They were a mixed lot, rich and poor, humble and great. He would entertain them whilst continuing with whatever was the task in hand, the while smoking a pipe which had been burnt away by constant lighting at a neighbouring bunsen. His most recent assistant would be introduced to visitors of world renown. It is doubtful if any other person had such a galaxy of famous friends. He would arrive promptly at 10 a.m., come whistling up the stairs, pop round and pass a cheery word with all of us, and most probably relate the latest good story. Then to work, and at one o'clock lunch, which would be enlivened by anecdote, reminiscences and story. There were never vacant seats at his table. He took tea in the laboratory with his assistants. He insisted that we all possessed a distinctive and gaily coloured cup. Old assistants, now famous, would come, and it was great to see them bend under his shafts. But he loved them all, famous or otherwise. I honestly believe that he gloried in their successes more than in his own. At 4 p.m. he would depart to read in the libraries of the R.S.M., the Colleges or Patent Office until they closed. After a meal at home, there was more study until bedtime. He wasted no time and, being endowed with an astonishing memory, he became a veritable encyclopœdia. He always knew what had been done and what was being done. He amazed me with his knowledge of men and affairs in the veterinary profession. Many were the humorous stories he told of his experiences with our men. He was a severe critic who maintained a high standard of accuracy in his work, and demanded the same of others. He was the Macqueen of the London Hospital. His writings show that he was a master of good English. His handwriting was clear and distinct, and he invariably used a methyl violet ink.

He first came in contact with veterinary surgeons whilst at the Lister Institute. There he met Sidney Villar. He often told me how impressed he was by Villar's character and competence. It was fortunate that he first met such an able veterinarian. Bulloch never lost the high opinion which he then formed, and there is no doubt that it was largely due to Sidney Villar that he was always our champion. He held Sir John McFadyean in high esteem. He considered him to be an outstanding pathologist and a great teacher. He would advise his pupils to go to Camden Town to attend Sir John's lectures. On many occasions in conversation with me he would express his regret and disappointment that Sir John was not honoured by the Royal Society. He would express these views in no uncertain terms. For many years he examined in Pathology for the R.C.V.S. He had a high opinion of our students, and he stated in public that in his opinion their knowledge and understanding

of this important subject was of a higher standard than that exhibited by medical students. He was a good examiner, who could put a student at ease and so enable him to give of his best.

He was honoured by his University and by the Royal Society. He served on many committees, including that dealing with foot and mouth disease. He would get to know personally all those with whom he came in contact, however humble their station, and he never forgot them. A kind word here and a jest there made him the friend of all.

He married in 1923, and the years which followed were blest by great happiness, and saw a mellowing of the old pugnacious spirit. This was evident to those who had the privilege of visiting his home. Unfortunately, the last few years were handicapped by a gradually progressive paralysis agitans, but he did not allow it to interfere with his work. Mentally, he was as good as ever, and even as late as 1939 learned Italian in order to progress better with a work upon Spallanzani.

He was compelled to enter his old hospital as a patient on February 9th, and died on the 11th.

We mourn a great scientist who was a kind and generous man and a staunch friend and admirer of our profession.

J. McC.

Editorial

FOOT AND MOUTH DISEASE

It will be seen from the public Press that outbreaks of foot-and-mouth disease have occurred in various parts of the country, and that the Minister of Agriculture has made an Order applying the provisions of the Foot-and-Mouth Disease (Controlled Areas Restrictions) General Order of 1938 to a number of counties. It is pointed out that the application of this Order is "designed to control the movement of animals . . ." and that "it is a purely precautionary measure to prevent the spread of disease to other parts of the country, and was necessary because animals affected by the disease have been widely distributed in the area in question from a market in that area." It is of the utmost importance to-day that foot-and-mouth disease is kept under control, and it is therefore pleasing to know that the authorities are adopting all necessary precautions to prevent its spread. It will also have been observed in the Press that the age-old subject of the slaughter policy in dealing with foot-and-mouth disease control has again been criticised, and that in reply letters have appeared from the Minister of Agriculture and Fisheries and from Sir Joseph A. Arkwright, Chairman of the Foot-and-Mouth Disease Research Committee of the Ministry of Agriculture and Fisheries. The Minister deals effectively with much of the criticism and indicates clearly how the slaughter policy in this country has been effective and of low cost to the nation. He states that the average payments by the State in connection with the slaughter policy have averaged just over £100,000 for each of the last 15 years. He also makes clear, however, that the slaughter policy might have to be abandoned if the disease got out of hand in this country as it has in other parts of the world. There is little doubt that the slaughter policy must be continued as long as those responsible for the control of the disease are satisfied with its operation in the interests of the country. We look forward, when peace again prevails, to the livestock from this country being in demand for purposes of replenishment. Freedom from foot-and-mouth disease is accordingly of special consideration, and for this reason alone we trust that the Minister and his advisers will never find it necessary to adopt other methods for the control of the disease.

It has also been stated in the public Press that research into the causes and prevention of the disease has been neglected in this country or has been

done on wrong lines. Both the Minister and Sir Joseph Arkwright have replied effectively to such criticisms. The various Progress Reports of the Foot-and-Mouth Disease Research Committee bear ample evidence of the nature of the work carried out and the great advances that have been made concerning our knowledge of the virus, methods of spread, etc. Those who are unacquainted with or who have only a superficial knowledge of the disease as a whole may be apt to arrive at conclusions in their own minds concerning the methods they think should be adopted for the control of this highly infectious disease. Until its incidence becomes so great that the slaughter policy fails to control the disease, we earnestly hope that, in spite of criticism, it will be continued, especially in war time. It behoves all concerned, including veterinary surgeons, to assist the authorities in controlling the spread of the disease by reporting all suspicious cases at the earliest possible moment, not from fear of penalty inflicted on the owner because of failure to report, but from the national point of view of suppressing outbreaks in their earliest stages.

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General Articles

PUS—ITS SIGNIFICANCE TO THE PRACTITIONER

By W. M. SCOTT, F.R.C.V.S., D.M.V. (Zur.)
Bridgwater.

In this enlightened post-Victorian age many surgeons look upon pus as their *bête noire*—a by-product of inflammatory reaction and as such only to be treated as an undesirable quantum.

I believe that the old-time practitioners were more conservative in their outlook. They made a close study of all purulent discharges. They took a very critical interest in pus in order to gauge the progress or otherwise of their cases.

They recognised two kinds of pus, *laudable* or healthy pus, and unhealthy pus. When the former obtained they knew there was little or no cause for anxiety; *per contra,* if the latter was in evidence the patient's life was probably in jeopardy.

To obtain a clear understanding of the rôle pus plays in the destructive and constructive course of disease, one must have a working knowledge of the complex *causa causans* of pus production.

Inflammation

I do not like the term, but as it has been handed down for generations, to oust it now from our terminology would be difficult. On the receipt of an injury—mechanical, chemical, bacterial—the blood vessels contract, then immediately dilate, and the blood flow slows down. In the veins and capillaries many polymorphonuclear leucocytes, which in normal tissue are found in the centre of the stream, pass into the plasma zone and attach themselves to the endothelial wall.

By the aid of their pseudopodia they angle their way through the vessel wall and wander into adjacent tissue. The openings thus created prepare the way for the erythrocytes and the blood plasma, constituting the so-called inflammatory œdema. Coagulations follow and serum and fibrin are the result. If the injury to the tissue is gross, thrombosis occurs in the blood vessels and, also, in the lymphatics.

If the inflammatory irritation is due to pathogenic bacteria, the polymorphonuclears attack them and envelop them, i.e., phagocytosis. Later in the course of the disease mononuclear phagocytes devour the remaining undigested elements, which are carried to the nearest lymph nodes, where they are digested. In this defensive rôle, therefore, three phases are noted:

(i) Dilution by the body fluids.

(ii) Centralisation by the cellular elements.

(iii) Destruction of the offending "materiæ" with the removal of the irritant and cellular debris by the cellular and humeral elements.

The two great factors in the curative rôle in all inflammatory processes come, therefore, under two heads: cellular and humeral.

Cellular.—The phagocytes found mainly in inflammatory tissue are the polymorphonuclear neutrophiles, polymorphonuclear eosinophiles, lymphocytes, mononuclear wandering cells, and plasma cells.

The neutrophiles are most abundant when tissues are invaded by pyogenic bacteria, especially when the H-ion concentration of the exudate is 7 or above this point. These phagocytic cells and in pus are known as pus cells. When they die they liberate proteolytic ferments. These ferments exercise a powerful influence in the curative rôle, of which more will be discussed later.

The *eosinophiles* are found in chronic conditions such as actinomycosis and actinobacillosis, and are not so active as the former.

The *lymphocytes* are found most commonly in subacute and chronic inflammatory lesions. They do not appear to possess much proteolytic power.

The *mononuclear* phagocytes are often called the scavengers. They are present in such diseases as tuberculosis, botriomycosis, actinomycosis, actinobacillosis, glanders, and Johne's disease.

They appear at the end phase of disease after the H-ion concentration of the exudate falls below 7.0. In other cases, such as old-standing cases of actinomycosis, actinobacillosis and mastitis in bovines, after the polymorphonuclear elements have done their work and disappeared, *plasma cells* appear. Some authorities assert that they produce a proteolytic ferment, but in many cases this must be of a low and inconsistent order.

It is interesting to note here that the rise or fall of the H-ion concentration depends upon the types of cellular elements found in inflammatory discharges. Thus, if the H-ion concentration is between 6.8 and 7.0, macrophages—mononuclear phagocytes—predominate. If the rise is between 7.2 and 7.4, the predominating cells of the exudate are the polymorphonuclear leucocytes.

Humeral.—The inflammatory exudate is composed of blood plasma and material thrown out in the reactive process. It also exhibits immune bodies—opsonins, precipitins, bacterolysins, hæmolysins, and cytolysins. In the process

of coagulation, fibrin and lymph or serum are produced. This liquid content exercises a very potent effect on resolution by aiding phagocytosis, diluting soluble irritants, and maintaining normal H-ion concentration, but owing to tissue damage an interference with normal metabolism follows. As a result, the intake of nourishment and the output of metabolic waste become unbalanced.

The carbohydrate metabolism is upset, sugar decreases, lactic acid increases, and the alkaline reserve is depleted. A vicious circle of acidosis develops. Moreover, the fall of the H-ion concentrate reduces phagocytosis. Ferments now develop, which digest dead and dying cells.

The inflammatory exudates are variable, thus:

Serous—seen in mild forms.

Catarrhal—clear or viscous, seen mainly when mucous membranes are involved. This consists chiefly of lymph-carrying epithelial cells in various stages of degeneration, mucin, leucocytes and bacteria—active or dying.

Fibrinous—a jelly-like exudate from a serous or mucous membrane.

Hæmorrhagic—when the inflammatory process is violent and virulent, as, for example, in anthrax or quarter-evil.

Purulent—the breaking-up and liquefaction of dead tissue and the formation of fluid in variable density—constituting pus. Pus is made up of lymph, dead leucocytes, bacteria—dead or dying, tissue cells in various stages of life or death, and autolytic ferments. If these ferments are left unhindered, the digestion goes on, fresh tissue becomes invaded by pus, and abscess formation is the result.

In many cases an anti-ferment is produced, which controls the effect of the proteolytic ferments. An example of this is seen in tuberculosis where necrosis is much in evidence.

It will be gathered from the foregoing that the tissue reactions to irritation, especially bacterial, is a very complicated and involved one.

Pus Content

Inflammation—itself a complicated process—gives rise to pus, a complex compound. If a chemical substance, such as turpentine, clove oil, mercury or zinc salts, is injected into animal tissue, a fixation abscess develops. A positive chemiotactic attraction for polymorphonuclear leucocytes arises, culminating in pus formation. Such pus, however, is sterile, and carries with it small clinical risks. It is the simplest type of pus formation seen in practice, for it is composed of dead or dying tissue cells only, and is non-infective.

The type of pus of most interest to the practitioner, however, is impregnated with pathogenic bacteria, active, dying or dead. In a simple abscess so-called *laudable* pus has a specific gravity of 1030-1040, is thick, opaque, creamy

and whitish-yellow in colour. It contains about 10 to 15 per cent. of solid matter.

If placed in a test tube and allowed to stand, it separates into a dense yellow layer and a clear supernatant fluid.

Pus has an alkaline reaction, it contains proteins, peptones and other albumen-containing bodies, fatty acids, cholesterol and some pigment from the red blood corpuscles. There are also cells especially of the polymorphonuclear type undergoing digestive changes, some mononuclear cells, connective tissue cells, pathogenic bacteria, fibrous tissue, and peptonising ferments. It is dense and if placed between the finger and thumb exhibits a considerable degree of adhesive power.

Such a type of pus embedded in healthy tissue has no chance of " running riot," being controlled by the immunising resources of the body. The density, colour and consistence are due to the cellular content, and this in turn is due to the " battle royal " which has taken place between, on the one hand, the body cells, which have died in their onslaught on the bacteria, and, on the other hand, positive chemiotaxis. In the meantime, as a result of bacterial aggression, the tissue cells are activated and a fibroblastic wall surrounds the infected zone, preventing further extension.

Tryptic ferments at this stage come into play, and if left to nature the abscess points, and the content is evacuated. In some cases, however, the tissue cells—where the general health of the patient is below par—are inactive, the protective wall is weak, or it may be the ferments are particularly virile, or both those conditions may obtain and the pus runs riot, invading healthy tissue and endangering the life of the patient. Often at this stage an antitryptic ferment is produced, and if sufficiently strong, pus ramification is checked and life is not imperilled.

Now let us look at the other side of the picture where so-called unhealthy pus obtains.

It is an accepted maxim that the healthy intact mucosæ and epithelium of the macro-organism is immune to bacterial aggression if one may exclude such virulent infections as anthrax, glanders, blackleg, etc. Where, however, there is a breakdown of the integument or mucosæ, pathogenic bacteria gain a footing and may become exceedingly aggressive. In practice, one often sees abortive terminations following upon infection, largely due to the high state of the immunising rôle of the body, coupled, perhaps, with the low aggressive power of the attacking bacteria. If, however, the opposite obtains, then a serious state of affairs arises. The immunising mechanism breaks down, the bacterial aggression is on the ascendant, their activities reaching adjacent tissue with perhaps a break through to the circulation, culminating in a typical septicæmia, and often ending in dissolution. In such a case, let us presume the focus of infection is the distal end of the limb, marked œdema is noted, ex-

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tending up the limb to the groin or the armpit. When a poor defence is put up, the discharge from the wound is then watery and often sanious. This type of pus—so-called *unhealthy* pus—is greatly lacking in pus cells owing to the high virulence of the bacteria, they and their toxins have produced a condition known as *negative chemiotaxis*. That is to say, instead of the leucocytes and other tissue cells becoming attracted to the bacteria, they have been repelled and phagocytosis has failed, or partly so.

From this it will be seen what a valuable index pus is if the clinician will only take the trouble to interpret its meaning when assessing the course of the infection. It indicates Nature's reaction to the infection, and indicates to the practitioner how best he may employ his skill to assist towards resolution.

At this point it may be of interest to note two diseases in which pus formation is conspicuous by its absence, i.e., anthrax and quarter-evil.

In anthrax in cattle the alimentary mucosæ appears to put up a poor fight and a generalised septicæmia is soon in evidence. In the horse and the pig a better effort is indicated. The throat and its adjacent glands are the primary seats of infection. In this case a serous, followed by a serosanguineous discharge is the result, but phagocytosis is mainly of a weak character. In man often it manifests itself as a local infection, i.e., *malignant pustule,* and I think I am right in saying there is little or no purulent formation.

I remember when I was a student at college I developed a malignant boil on my neck below the right ear. It was painful and œdematous; a serosanguineous discharge followed, and eventually a heavy crust formed, but there was no pus formation so far as I can recollect. A college professor diagnosed the case as malignant pustule. It was presumed that I scratched my skin when dissecting the carcase of a horse which had died from anthrax. The authorities were not so particular about this disease in those days; in fact, I question if anthrax was a scheduled disease at that time. As in local pus formation, a similar reaction follows upon inflammatory diseases of the mucosæ in such systems as the respiratory, and the urino-genital. Here, too, the purulent content varies according to the virulence of the bacterial invasion on the one hand, and the cellular and humeral reactions on the other.

For example, there is a marked difference in the quality and consistence of the nasal discharge of glanders and that of strangles when the latter runs a normal course. While the former is grey, glairy, thin, lacking substance, deficient in cellular content, and sometimes blood-stained, the latter is creamy, thick and rich in cellular elements.

The virulence of the *Bacillus mallei* has been too much for the phagocytes —negative chemiotaxis is in evidence and bacterial action is on the ascendant. In strangles bacterial aggression is kept in check as a rule, and the purulent discharge is *laudable*. Where atypical or irregular strangles develops the immunological index has broken down owing, no doubt, to a lowered natural

vitality of the host. The purulent discharge is thin, grey and deficient in cellular content.

When the post-pregnant uterus is involved, great scope is found for bacterial ascendancy—a large amount of serum is thrown out from the uterine wall; the cellular elements, too, are very active in bringing about bacterial destruction, but the fact remains that there is ever an accumulation of inflammatory by-products, and by reason of the lack of drainage the whole organ becomes a veritable cesspool of corruption.

In the cow and the bitch the uterine wall and lymphatic system usually is capable of putting up a good defence against extension. The mare, however, is less fortunate, and septic metritis often culminates in pulmonary and laminitic sequelæ.

Why this is so it is difficult to explain, and if one reasoned anatomically, one would say the complex surface of the cow with its lacunæ, admirable for "housing" bacteria, would be more ideal for bacterial growth than the more simple surface of the mare. Be this as it may, the purulent elements are usually thin, watery and serosanguineous, showing a low phagocytic power of the uterine mucosæ, with thrombosis of the capillaries to aggravate and involve matters.

Therapeusis

From the practitioner's point of view, the foregoing observations may seem largely academical, but I venture to think these findings, if kept in view, will materially assist us by showing how Nature becomes the Master Physician, and enable us to help her in the curative rôle.

In those cases where *laudable* pus is in evidence, danger to life is minimal and needs little attention on the part of the practitioner; *per contra*, where it is unhealthy, our efforts must be skilful, energetic and untiring: where there is infection through the skin with a spreading œdema, lancing is indicated to relieve vascular tension, followed by hot fomentations, to which is added a *mild* antiseptic and common salt. Constitutional disturbance, if present, must be treated accordingly. A bacteriological examination of the pus to ascertain the type of organism present is most helpful. The appropriate antisera may be injected. This should be followed by a vaccine—preferably an autogenous one to activate the immunological forces.

As regards the treatment of the infected wound discharging *unhealthy* pus, it must be our endeavour to stimulate phagocytosis and so hasten the production of *laudable* pus. If the case is one of some standing, the walls may have become coated with impervious fibrin, necrotic tissue, dead and dying cells and active bacteria securely "housed" against the action of the phagocytes and the bacteriotropic serum. Thorough curetting is then indicated, after which the wound should be irrigated with a one per cent. solution of sodium citrate mixed with normal saline solution at blood heat. The former

acts as a decalcifying agent, reducing the coagulation point; the latter, by its osmotic power, "draws" out the bacteriotropic serum. To activate phagocytosis I know of no better dressing than gauze soaked in the old-fashioned preparation—turpentine and creosote, in rape or olive oil.

On no account must strong antiseptics be used; they do not destroy the bacteria—many of which, as already stated, are securely "housed" deep in the tissues and coagulated exudate. They thwart Nature's effort, which, generally speaking, if given a sporting chance, is capable of dealing with the aggressors. It may be urged that creosote is a powerful and irritant antiseptic, but the author of this formula wisely "buffered" this agent by adding an oleaginous preparation, and as this was probably long before phagocytosis was ever thought of, we see here another illustration of practice preceding science. Bier's treatment in selected cases is often beneficial, producing a beneficial hyperæmia and stimulating the body forces to surround and destroy the foci of infection.

Where one is dealing with *unhealthy* purulent discharges from viscera, the first objective of the practitioner is to raise the immunising index by the administration of suitable foodstuffs. If the organs of assimilation are below par, it is obvious that Nature will not make the most of the food content until digestion is improved.

Just as the digestive system must have good food, so, too, must the lungs have good air in order that a high degree of blood oxidisation may be maintained. Then again, owing to metabolic changes, effete material is being continually manufactured and must be thrown off through the three channels, skin, bowels, kidneys. It is essential that these structures be kept in an efficient working state. The specific mode of treatment consists of (a) biological—sera, vaccines; (b) biochemical—of this there is a large group which, in the light of our present knowledge, is headed by agents of the sulphonamide group.

Summary

I have endeavoured to show that there are two definite types of pus met with in general practice, i.e., laudable and unhealthy; and that where these are recognised the expert can be greatly assisted in the fields of prognosis, diagnosis and therapy.

Where laudable pus is in evidence, there is, as a rule, no serious danger to life. Unhealthy pus, however, must ever carry with it an element of suspicion and concern, and a menace to life should extension take place.

In the treatment of pus formation, drastic and heroic measures are generally contraindicated. One must ever remember that Nature is the Master Healer, and be ready to react to the signals she displays to those who have eyes to see.

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FIG. 1.—Cross section of muscle showing four larger and some small lesions.

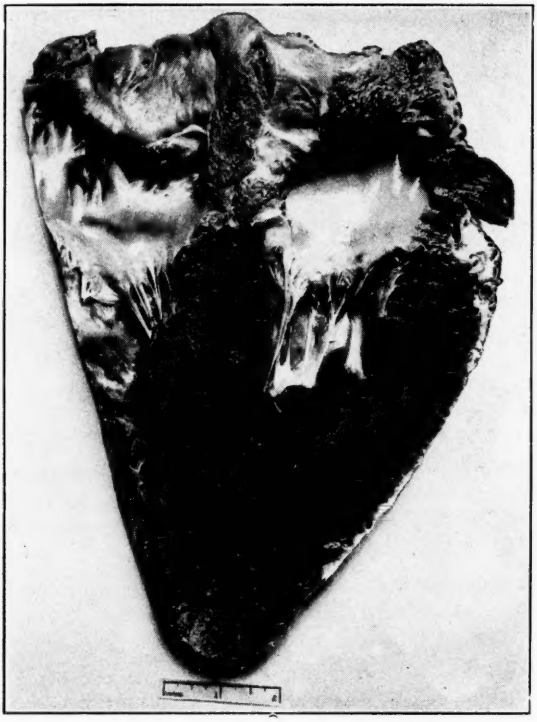

FIG. 2.—Heart wall showing the small thickened arteries in the myocardium.

PERIARTERITIS NODOSA IN CATTLE

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THE term, periarteritis nodosa, has been applied in human pathology to a rare condition in which small inflammatory nodules develop along the course of the arteries in various parts of the body. This disease primarily affects the tunica adventitia, but the tunica media and intima also become involved. They become infiltrated with leucocytes and there is a tendency to the formation of small aneurysms.

In animals, the name has also been applied to nodular or elongated thickenings of the vessels, especially the middle-sized and smaller arteries in connection with the muscles and various organs. Cases of this nature have been described in cattle, pigs, a dog and an antelope. The etiology of the affection has not been ascertained, and it is not improbable that the cause is not the same for each of the species of animals in which the disease has been described.

Although we have not come across records of the kind in this country, we are inclined to believe that the disease is not rare in bovines. We have two specimens in our collection, and have seen a number of others, and recently another case has been brought to our notice.

The cattle affected have been slaughtered at the abattoir, and the affection only noted when the musculature and some of the organs were examined by the meat inspector. They appear to have been all Irish cattle, and our last specimen was received from Mr. Clayton, M.R.C.V.S., of Birkenhead. The animals were in good condition, one was a heifer of about two years old, and two others about three years old. There was no evidence that any symptoms of ill-health had been shown prior to slaughter.

In the last case examined lesions were seen in the general musculature, in the myocardium and the subepicardial fat, as well as in the mammary gland, but in some previous cases lesions were also noted in the spleen and cortex of the kidneys, liver, lungs, the musculature of the back and thighs, the tongue and diaphragm. The aorta and larger vessels were normal. (See photographs 1, 2, 3 and 4.)

At first sight the lesions in the musculature appeared to resemble the macroscopic calcified cysts occasionally seen in sarcosporidiosis, but necrosis and calcification were absent. They consisted of little grey areas, dotted through the muscles and organs, many of them showing the open mouths of the affected vessels. In cross-section they varied in width from a pinhead to a barley grain, and were irregularly oval or circular in outline. For the most

part the lesions did not consist of distinct nodules, and this was particularly evident in the adipose tissue. They consisted of considerable tracts of somewhat tortuous vessels with greatly thickened walls. The affection was confined to the medium-sized and small arteries.

On microscopical examination of sections from the affected parts, the changes were often observed in six or seven vessels within a square inch of tissue. In some cases the vessels were cut at several points, so that on account of their tortuous course more than one vessel appeared to be involved, and in addition small side branches were also sectioned. They were surrounded with thick strands of fibrous tissue arranged for the most part in concentric layers round the vessel walls, and in advanced cases this fibrous tissue had invaded the tunica media and tunica intima. The thickening at all points was symmetrical, and in the media as well as in the adventitia the strands of collagen fibres formed concentric layers. In the former there was some atrophy of the circular non-striped muscle fibres. In the tunica intima the new tissue was highly cellular, fibroblast cells being abundant and collagen fibres sometimes absent or sparse. (See photomicrograph 5.)

This new formation accounted for the greatly increased thickness of the vessel walls, and caused a great diminution in their lumen. Indeed, in some instances the lumen had been obliterated, only some straggling red blood corpuscles being found among the cellular elements of the tunica intima. In the latter mass also new capillaries had often formed in the granulation tissue in the centre of the obliterated arteries.

From what has been stated, the lesions might be more accurately described as a periarteritis combined with an endarteritis obliterans, and the changes presented were similar to those described in the vessel walls in the gummata of human syphilis.

In the new fibrous tissue surrounding the affected arteries small vessels like vasa vasorum were very prominent and numerous as if they had undergone some compensatory hypertrophy, to make good the damage done to the parent vessels.

Numerous eosinophiles together with some polymorphonuclear leucocytes were present in the tunica adentitia, and some eosinophiles were found between the cells of the tunica intima and even in the lumen of the vessels.

Moreover, at some points near the lesions there were small masses of brown pigment granules, many of which were deposited in the bodies of the histiocytes. These granules proved to be granules of hæmosiderin and stained blue with potassium ferrocyanide and hydrochloric acid.

The precise cause of the condition was not apparent. In various parts of the muscles there was a light infestation with sarcosporidia, but the latter had no contact or relation to the vessel walls, and could only be regarded as incidental or as complications. Occasional muscle fibres, single or in small

FIGS. 3 and 4.—Kidney showing lesions in cortex and boundary zones, and cross-section of spleen.

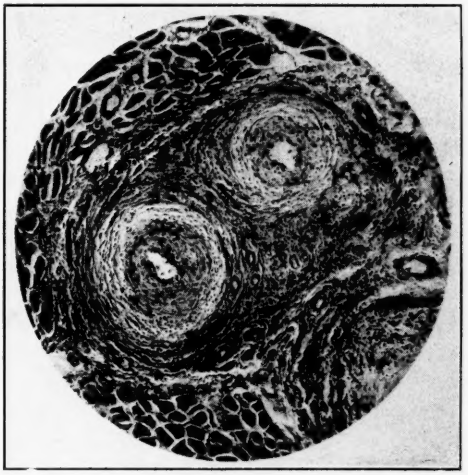

FIG. 5.—Photomicrograph showing the changes in cross-section of two adjacent arteries. X 60.

groups, throughout the muscle sections presented evidence of hyaline degeneration, which was no doubt dependent upon interference with the blood supply through the decrease in the lumen of the nutrient arteries.

The presence of brown granules in the neighbourhood of the affected areas pointed to the remains of some local damage and hæmorrhage, and the presence of the eosinophilous corpuscles would lead one to suspect that at one time animal parasites such as helminths may have wandered out from their usual situations and set up the changes in the vessel walls. The changes there would therefore be regarded as attempts at repair. This seems to be a reasonable explanation in view of the frequency of helminth affections of the alimentary canal and respiratory system in young cattle.

The chief interest in the cases described lies with the meat inspector, in the recognition of the changes which are to be found in the course of his duties during the examination of carcases.

REFERENCES

Nieberle and Cohrs: Lehrbuch der Speziellen Pathologischen Anatomie der Haustiere, 1931, p. 50.
Hutyra, Marek and Manninger: Special Pathology and Therapeutics of the Diseases of Domestic Animals, 4th Edition, 1938, Vol. II, p. 680.
Boyd: Textbook of Pathology, 1938, p. 436.
Ostertag: Text of Meat Inspection—Young, 1934, p. 177.

JOHNE'S DISEASE INFECTION OF LABORATORY ANIMALS

(*Concluded*)

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In a previous paper (1940) the author reported the results of certain experiments on guinea-pigs (and other small animals), using liquid paraffin emulsions of Johne's bacilli, on the lines reported earlier by Mohler (1939). These showed that while it was easy to set up lesions in the guinea-pigs similar to those described by Mohler by the intraperitoneal inoculation of liquid paraffin emulsions of Johne's bacilli, contrary to his claim, the inoculated guinea-pigs did not always die but continued to live for prolonged periods, though extensively diseased.

The author was also unable to obtain any positive results from his attempts to transmit the infection from one guinea-pig to another, either by using liquid paraffin emulsions or saline suspensions of lesions. Slight lesions were met with, generally in the omentum, but sometimes also in the liver,

which were in conformity with the small number of bacilli present in the injected material.

Cultural examination of material from some of the guinea-pigs showed that the bacilli were gradually killed off in the body of the guinea-pig and that the lesions were only a response on the part of the tissues to resistant foreign material in the form of acid-fast bacilli. There was no active multiplication and no progressive disease.

Since the cultures used in the experiments described in that paper were rather old, two to three months, it was thought that such differences as were noted by the author between his results and those of Mohler might be due to the majority of the bacilli being either dead or in an inactive state of multiplication. Experiments were therefore carried out using young cultures of the three strains used in the previous series, together with another—Weybridge 97.

Experiment I

With a liquid paraffin emulsion of a 17-day-old culture of Weybridge Strain 2, three guinea-pigs were inoculated intraperitoneally, receiving 1, 5 and 15 mgms. of the culture respectively. All the guinea-pigs survived, and when killed after three months, showed the usual type of lesions, including small translucent foci in the lungs. Such foci have been described by Hagan and Levine (1932), but were not seen in the guinea-pigs inoculated in the previous series.

Experiment II

From a 17-day-old culture of the Royal Veterinary College strain used in Experiments I and II previously, a liquid paraffin emulsion was prepared, and with this three guinea-pigs were injected, receiving 1, 4 and 7.5 mgms. of the culture respectively. The 1 and 4 mgm. guinea-pigs died after about three weeks. The 7.5 mgm. guinea-pig survived and was killed after seven weeks, and from this, four guinea-pigs were inoculated, two with a saline suspension of the nodules and two with a liquid paraffin emulsion. The material previous to inoculation showed large numbers of strongly acid-fast bacilli, but direct cultures on a series of tubes without treatment with antiformin or other chemicals showed no growth up to six weeks.

All these four guinea-pigs remained perfectly healthy and were killed after six weeks. The saline suspension guinea-pigs showed only a few nodules in the omentum, while the liquid paraffin emulsion guinea-pigs showed, in addition, lesions on the diaphragm.

Experiment III

An emulsion was prepared from an 18-day-old culture of the strain isolated by the author and previously used in Expt. IV. With this, three

guinea-pigs were inoculated, receiving 0.5, 5 and 10 mgms. of culture respectively.

All three guinea-pigs died in about three weeks.

Experiment IV

With a liquid paraffin emulsion of an 18-day-old culture of Weybridge Strain 97, three guinea-pigs were inoculated, receiving respectively 1, 3 and 10 mgms. of culture. After four weeks all three were tested intradermally with four separate injections of Johnin and avian tuberculin in dilutions of 1 : 10 and 1 : 50, following which the 10 mgm. one died.

The others continued to live, and were destroyed after three months.

Post-mortem examination, besides other lesions, showed a few small translucent foci like miliary tubercles in the lungs.

It will thus be seen that of the total number of 12 guinea-pigs inoculated with the four different strains, five died within a period of three weeks and one died following testing with Johnin and avian tuberculin. The others continued to live for several weeks in perfect health, though when killed all showed well-marked lesions of the type described by Mohler and in the author's previous article. The percentage of deaths was thus higher than in the previous series, but it does not invalidate the general conclusion that quite a large number of the inoculated guinea-pigs survive such infection, especially if in good health and condition, for several months. Moreover, as will be seen from the results of the above experiments, there is no correlation between a fatal termination and the dose. Sometimes, while guinea-pigs receiving a smaller dose die, those receiving larger doses survive. Furthermore, as has been shown previously, similar fatal results can be produced by other acid-fast organisms, including dead pathogenic and live or dead saprophytic organisms.

Cultures were made from only one guinea-pig seven weeks after inoculation, but no growth was obtained, which again supports the author's previous conclusion that there is no active multiplication of the bacilli, and that no progressive disease is set up.

Transmission experiments with a saline suspension or liquid paraffin emulsion of nodules gave no better results than in the previous series. A few lesions were, of course, found and these were perhaps slightly more generalised than those following injection of plain saline suspensions of culture, but this can be easily explained by the fact that in the former case the bacilli are enclosed in and protected by particles of tissue, and have thus a better chance of being held up and withstanding the defensive mechanism of the body and of producing lesions.

Incidentally, it may be stated that most of the above guinea-pigs were tested at various times by the intradermal injection of dilutions of avian tuberculin and Johnin, and they all reacted strongly to both, though there is some evidence that the sensitivity after some months was on the wane, correlated

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probably with the completion of the destruction of the bacilli in the tissues.

Similarly, guinea-pigs injected with a saline suspension or liquid paraffin emulsion of nodules from a previously infected case also reacted, and in one case, where two such guinea-pigs from the previous experiment had survived, reactions were obtained after as long as 4½ months from the date of inoculation. The reactions were, however, mild, and the allergy appeared to be disappearing.

REFERENCES

Hagan, A. William, and Levine, Philip: (1932) *J. Amer. Vet. Med. Assoc.*, N.S., *34*, 723.
Mohler, William M.: (1939) *Ibid.*, N.S., *47*, 590.
Sahai, L.: (1940) *Vet. J.*, *96*, 407.

Clinical Articles

TWO CASES OF PROLONGED ARTIFICIAL RESPIRATION

By G. H. ARTHUR
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CASE I

Subject

A TWO-YEAR-OLD black and white terrier, weighing 25 lb.

History

The animal had sustained a compound fracture of the left forearm.

It was intended to place the patient under light nembutal anæsthesia and to apply a windowed cast.

Operation

As usual, the calculated dose of nembutal (gr. 5) was placed in the syringe, the first two-thirds of the dose being fairly quickly injected, and then small amounts injected cautiously at intervals of half a minute, until the requisite degree of anæsthesia was present. After giving the whole dose, there was still some slight mandibular resistance, and the pedal reflex was present but much diminished in degree. Considering the nature of the surgical interference, anæsthesia was thought to be sufficiently deep. A plaster cast was about to be applied when, two minutes after completing the injection, respirations

ceased. Artificial respiration was commenced immediately. The heart's action remained good, but natural respirations could not be induced until 63 minutes after injecting the nembutal.

A cast was then applied. No further incidents occurred and the dog made a good recovery.

CASE II

Subject

A four-year-old fox terrier, weighing 21 lb.

History

There had been inveterate shaking of the head for some weeks, but the dog had not responded to the usual treatment for otorrhœa.

On examining the ears with the auroscope, a deep-seated otitis was noted. It was decided to perform aural resection on both ears.

Operation

After injecting gr. 4 nembutal, there was satisfactory anæsthesia; mandibular resistance was overcome, but there was still a well-marked response to pinching the web of the foot. The operations proceeded satisfactorily, but after 30 minutes respirations ceased. Artificial respiration was commenced and had to be continued for a period of 70 minutes, at the end of which automatic breathing recommenced. The heart beat regularly throughout. Approximately 45 minutes after resorting to artificial respiration the dog evacuated its bladder.

The animal recovered normally and appeared none the worse for his experience.

Discussion

The particular interest of both these cases was the prolonged period during which normal respiratory activity was in abeyance. Little literature has appeared on this subject in veterinary practice, but McCunn[1] has recorded a recovery after 35 minutes of artificial respiration. In each of the above cases the dog was placed on its side with its head overhanging the table edge. It was found that the resiliency of a rubber cushion (placed under the patient) aided the respiratory movements and helped to prevent damage from the manual compressions of the chest. These compressions should be made to resemble the normal respiratory movements and frequency as closely as possible. At intervals of 10 minutes the animal was turned on to the other side.

The writer has little respect for chemical stimulants in respiratory failure due to nembutal. It would appear that in such cases one has to continue artificial respiration until a sufficient amount of the drug has been eliminated to enable the respiratory centre to resume its function.

REFERENCES

(1) McCunn: "Hobday's Surgical Diseases of the Dog and Cat."

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PROLAPSE OF THE UTERINE CERVIX IN A COW

By C. WEIGHTON, M.R.C.V.S.
Lincoln.

Subject

A LINCOLN Red Shorthorn pedigree cow, about nine years old; in good condition.

History

The cow had produced several calves, had not previously been ill, and was due to calve at the end of December.

Examination, 25/12/40

When first seen the cow was found to be straining considerably, and an enlarged cervix was being protruded completely through the vulva each time the cow strained. The central annular ring of the cervix was about six inches longer than the average normal structure.

For a few days prior to my visit partial prolapse had taken place at intervals, and the cervix usually showed when the cow was lying. The animal was otherwise in normal health, and there was no elevation of temperature. No prolapse of the vaginal wall was seen as in the more usual type of case met with, except the circular portion at its junction to the cervix.

Diagnosis

The cause of the condition was believed to be due to inflammation and hypertrophy of the cervix, involving particularly the central annular ring.

Prognosis

The owner was informed that after calving the symptoms would probably improve, but the case was pointed out to be serious.

Treatment

The vagina was irrigated with a weak solution of *hewsol* and the vulva sutured with tape, making a single Halsted suture.

26/12/40.—Condition similar but the temperature was 103.6 F., and frequent attempts at straining continued.

28/12/40.—Condition more satisfactory. The vagina and vulva were again cleansed and a long piece of tape was attached to the first suture and pulled through to replace the first. The tape was tied in a large bow so that it could be loosened far enough to allow the cow to calve without having to be removed and tied again after parturition.

29/12/40.—The cow calved a normal bull-calf, and the owner retied the tape so that, in the event of the uterus protruding, a barrier would be met to prevent a rapid prolapse, and the same measure of control would be maintained over the cervix should the former not occur.

31/12/40.—On revisiting the farm the cow was found to be down with Milk Fever. A satisfactory response was obtained in about four hours to a subcutaneous injection of calcium boro-gluconate.

The afterbirth had come away by this date, the owner reporting that this was expelled about 24 hours after calving without the tape materially interrupting its passage. Only one part of the tape was found in position. It was holding at two points only, one on each side of the vulva. The tape was reinserted to complete the Halsted suture as originally fixed.

8/1/41.—Frequent straining with partial prolapse during the preceding three or four days had resulted in the cervix becoming gangrenous in parts.

The hypertrophied part of the cervix, about six inches long and four inches in diameter, was ligatured with elastic so that the greater portion of the hypertrophied ring would be removed, and acriflavine emulsion applied.

The cow having been removed to a stall from the loose-box where she calved, the hind quarters were raised with wet bedding to more than one foot above the normal level.

11/1/41.—Systemic disturbance with elevated temperature. Fever and stimulant medicine prescribed. An additional ligature was applied under spinal anæsthesia, the vagina irrigated and the same non-irritant antiseptic emulsion applied. Chloral hydrate draughts were left with the owner to be given should there be undue pain and straining.

13/1/41.—By this date considerable loss of condition had occurred. There had been straining at frequent intervals, and the temperature was 104.4 F.

The ligatured portion was removed without hæmorrhage, the stump dressed, and the ligatures left to slough off.

15/1/41.—Condition more satisfactory. Temperature 103.6 F.

28/1/41.—A most decided improvement was exhibited; no straining had occurred for about a week, and the cow had begun to put on weight. The suture material had by this time come away, owing perhaps to the action of the urine.

21/2/41.—Inquiries by telephone revealed that satisfactory progress was being maintained.

Discussion

The result may have been favoured by the gradual involution of the uterus but, in view of the absence of any abnormality of adjoining organs, which might give rise to similar symptoms, the success is attributed to the removal of the greater portion of the inflamed tissue responsible, together with local antiseptic treatment.

XUM

Correspondence

FOOT-AND-MOUTH DISEASE

CONTROL POLICY

Following correspondence in several newspapers criticising the official slaughter policy in the control of foot-and-mouth disease in this country, replies from the Rt. Hon. R. S. Hudson, Minister of Agriculture and Fisheries, and Sir Joseph A. Arkwright Chairman of the Foot-and-Mouth Disease Research Committee of the Ministry of Agriculture and Fisheries were published. They are reproduced below.

From *The Sunday Times.*

From the Rt. Hon. R. S. Hudson, Minister of Agriculture and Fisheries.

Sir,—The letters which have appeared recently in the Press directed against the official slaughter policy for foot-and-mouth disease call for a brief statement to correct many false impressions which those letters may have created in the public mind. It would be impossible to deal in a letter with all the points advanced by the critics of the policy, and I will, therefore, confine myself to the more important considerations as I see them.

(1) It is not suggested that foot-and-mouth disease is a "killer"; for the most part, animals recover naturally and without the assistance of any cure, but admittedly it reduces the milk supply considerably and affects the output of meat owing to the general loss of condition in adult stock; in very young stock the mortality is frequently high. The chief of the German veterinary service estimated that the severe outbreaks in 1937-38 in that country cost German farmers £83,000,000.

The Slaughter Policy

(2) The slaughter policy is not peculiar to this country. It is the policy of the U.S.A., the Union of South Africa, Northern Ireland, Eire, Norway, and Sweden, and is also followed by Switzerland unless the disease gets beyond control. The countries that "let the disease rip," to use Lord Bledisloe's words, only do so because they are unable to control the disease and not as a deliberate policy. International scientific veterinary opinion very strongly supports a policy of slaughter as opposed to any other method of dealing with outbreaks wherever circumstances render such a policy practicable.

(3) There is always the risk that the disease may get out of hand in this country also. In that case the slaughter policy would have to be abandoned. In the meantime payments by the State in connection with the slaughter policy have averaged just over £100,000 for each of the last 15 years. Can anyone

who realises the consequences of the abandonment of the slaughter policy contend that this is an excessive premium to pay against the disease becoming endemic, having regard to the value of the livestock of the country?

Committee's Report

(4) Early in 1924 a Departmental Committee was set up to consider the very heavy and extensive series of outbreaks of the preceding months and to review and report upon the slaughter policy. After a thorough investigation of the slaughter versus isolation issue, the committee strongly and unanimously recommended the continuance of the slaughter policy.

(5) Informed farming opinion generally whole-heartedly supports the slaughter policy. In a letter on the subject which appeared in a recent issue of one of the leading agricultural journals, the chairman of the Animal Diseases Committee of the National Farmers' Union stated that " slaughter is emphatically the only policy for this country."

Highly Contagious

(6) Foot-and-mouth is probably the most contagious disease known either in veterinary or human medicine and, contrary to what has been suggested, there is overwhelming evidence that the condition of the animal and of the soil from which it derives its food and the methods of husbandry practised make no difference to its susceptibility to the disease or to its powers of resistance. It is the highly contagious nature of the disease that calls for the utmost rapidity in slaughtering of affected stock, or stock which has been exposed to infection, so as to prevent its spread, since every affected animal remains an active manufacturer of the virus, and owing to its extreme diffusibility there is not the slightest hope of confining an isolation experiment, which has been suggested in one quarter, to any particular part of the country. The reason for this is that infection can be carried mechanically through the medium of persons, birds, rabbits, foxes, etc., or any article which has been in contact with diseased stock.

Losses Since the War

(7) In answer to the contention that the official policy leads to a large and unjustifiable waste of food in war time, I wish to make it clear that since the outbreak of war only the carcases of diseased animals have been destroyed. Contacts are salvaged for human food. There have been 237 outbreaks of the disease since the beginning of 1940, in the course of which the carcases of 3,098 cattle, 3,139 sheep, and 1,725 pigs have been destroyed. The remainder of the meat was salvaged for human consumption. The loss of meat and milk to the nation would undoubtedly have been very many times greater if the opponents of the slaughter policy had had their way, and if as a consequence the disease had run riot throughout the country.

(8) Finally, the suggestion that blind adherence to the slaughter policy

has led to neglect of research and experiment is devoid of truth. For many years research has been conducted on a substantial scale at a station devoted solely to the problems of foot-and-mouth disease but, unfortunately, no satisfactory alternative method of coping with the disease has yet been discovered by research in this or any other country.

R. S. HUDSON.

Ministry of Agriculture and Fisheries, S.W.1.

From *The Times.*

In the course of a communication on the above subject in a recent issue of *The Times,* Sir Joseph A. Arkwright, Chairman of the Foot-and-Mouth Disease Research Committee of the Ministry of Agriculture and Fisheries, observes: "The recent letters appearing in *The Times* and elsewhere condemning the slaughter policy for dealing with foot-and-mouth disease have relied on two fallacies: (1) That slaughter has in the past not prevented extension of the disease nor reduced the resulting public expenditure; (2) That scientific research has not been actively carried on in this country or has been on wrong lines.

"Regarding the first, the cost to the public and herd-owners in countries where reliance has been placed on isolation and treatment has been shown repeatedly to be far greater than in this country. Account must be taken of the almost universal spread of epidemics and of endemic persistence when attempts are made to combat the disease by isolation, as has been done in France and Holland.

"In the second place, scientific research has been continuously carried on, and resulting advance in knowledge of the disease has been considerable, as is evident from the contents of the five progress reports of the Foot-and-Mouth Disease Research Committee. This is acknowledged, but Dr. Crofton would prefer that all the exact and detailed knowledge of virus diseases, including foot-and-mouth disease, should be ignored in favour of a doctrine which he advocates, but which in the case of foot-and-mouth disease has been repeatedly disproved and rests purely on unsupported assertions, and is, moreover, at variance with all accepted scientific opinion. The Research Committee has examined such evidence as Dr. Crofton has been able to produce and found it quite unconvincing. Opposed to his statement that he has never been given any opportunity in this country to prove his thesis that a bacillus is of importance in the causation of foot-and-mouth disease is the fact that in 1939 he was given facilities for culture of his bacillus from foot-and-mouth disease lymph and its inoculation into guinea-pigs in accordance with his own proposals. The experiment was a complete failure, the results of inoculation being entirely negative both as regards infection and protection."